Cardiac Actinomycosis
A Case Report and Survey of the Literature

By Samuel J. ZOECKLER, M. D.

Involvement of the heart by actinomyces is rare. A case of actinomycosis, with involvement of the chest wall and clinical pericarditis, with recovery, is reported. The organism, Actinomyces bovis, was isolated from the lesion and accurately identified. The literature is reviewed and the modern concepts of pathogenesis and therapy are discussed.

In 1877 Bollinger described actinomycosis in animals. Wolff and Israel, in 1891, isolated the "Ray Fungus" from a human case. Since then there have been numerous reports of actinomycotic infections in man,1 and also a few cases of actinomycotic heart disease. However, no case of clinical pericarditis due to Actinomyces bovis was found in the available literature. The purpose of this paper is to report such a case and to present some modern concepts of the pathogenesis and treatment of actinomycosis.

Case Report

L. S., a 41 year old white male farmer, was admitted to the hospital June 24, 1950. Two months before admission he noted a pea sized, nontender, nonfluctuant nodule beneath the skin over the right sternal border. This gradually enlarged. Four weeks before entering the hospital he observed that the overlying skin became purplish-blue in color. During this period he had three episodes of severe, stabbing substernal pain. The first, two months prior to admission, lasted 12 hours; the second, one month before admission, lasted 20 minutes; and the third, two days prior to admission, lasted 10 hours. The pain did not radiate but was accentuated by deep inspiration and relieved by assuming the sitting position. All attacks began in the evening while the patient was at rest and were not associated with exertion. Between the acute episodes he noted transient, mild, substernal pain radiating to both shoulders. In the two month period there was a weight loss of 20 pounds.

In 1944 the patient was operated upon for appendicitis. A draining sinus developed at the operative site from which a yellow, serous material exuded with permanent closure after six months.

Physical Examination. The patient was a well developed, well nourished white male of florid complexion, having no obvious distress. Temperature was 100.8 F. Numerous carious teeth were present. A firm subcutaneous mass, measuring 4 by 2 by 2 cm., was found at the level of the seventh rib, extending to the right sternal border. It was attached to skin and deeper structures. The overlying skin had a violaceous hue. The mass was neither tender nor fluctuant. Physical examination of the lungs and abdomen revealed no significant abnormalities. A loud pericardial friction rub was heard over the entire precordium and posteriorly at the angle of the left scapula. It was loudest in the left third intercostal space, and was present during voluntary apnea. The heart was not enlarged and a sinus rhythm was present. Blood pressure was 110/70. The normal heart sounds were obscured by the friction rub.

Laboratory Findings. Examination of the blood revealed a white cell count of 8,300 with a normal differential. Red blood count was 4,170,000 with 13.5 Gm. hemoglobin and a hematocrit of 42 per cent. Urinalysis was normal. Typhoid and paratyphoid agglutinations were compatible with previous immunization. An x-ray film of the chest showed prominence of the right hilar markings with some thickening of the pleura on the right above the cardiophrenic angle. There was a soft tissue swelling at the level of the sixth and seventh ribs anteriorly on the right. An electrocardiogram showed slight elevation of the S-T segments in leads I, II and III (fig. 1). Tuberculous pericarditis with involvement of the chest wall was considered. Repeated smears and culture of sputum and gastric content were negative for acid fast bacilli. A skin test with second strength purified protein derivatives was positive.

A Silverman needle biopsy of the mass on the anterior chest wall was obtained on the tenth hospital day and a simple aspiration was performed on the eighteenth hospital day. Both specimens revealed Actinomyces bovis. The organism was anaerobic, non-acid fast and gram positive. Its identity was
confirmed by the laboratories of the United States Public Health Service.

Hospital Course. On the third hospital day the patient was placed on 300,000 units daily of procaine penicillin G in aqueous suspension and received a total of 8,700,000 units. On this regimen the temperature, which had been elevated since admission, became normal on the twelfth hospital day and remained normal throughout his hospital course. The pericardial friction rub was not audible after the sixteenth hospital day. Aureomycin was begun on the thirty-second hospital day, 0.5 Gm. every six hours for a total of 26.0 Gm. On the thirty-third hospital day the lesion on the chest was again aspirated. Smears and culture of the material were sterile. An electrocardiogram on the sixteenth hospital day showed inverted T waves in leads I, II, aV,

Figu1. Electrocardiograms taken at representative periods during the patient's illness demonstrating the changing electrocardiographic picture compatible with pericarditis.

sterile. An electrocardiogram on the sixteenth hospital day showed inverted T waves in leads I, II, aV₃, V₄, V₅, and V₆, and 14 days later continued to show changes compatible with "chronic pericarditis." (fig. 1). The mass slowly resolved and at the time of discharge was present only as an area of slight induration beneath the skin. Two teeth were found to have "apical abscesses." These were removed and cultured but no fungi were recovered. The heart was clinically normal at the time of discharge. Chest x-rays taken on discharge revealed no abnormality in the posteroanterior film. The lateral view demonstrated clearing of the inflammatory process. The patient was discharged on the forty-ninth hospital day. The iodides were not used in treatment.

Examination three months after discharge revealed normal physical findings, a normal electrocardiogram and an unchanged roentgenogram of the chest.

Discussion

It is believed that this represents the first reported case of actinomycosis of the chest wall associated clinical pericarditis. The organism Actinomyces bovis was isolated from samples aspirated from the patient's chest lesion and was accurately identified.

Other cases of actinomycosis involving the heart have been reported. Sanford and Voelker in reviewing 670 cases of actinomycosis found no cardiac involvement. Kaspar and Pinner found cardiac involvement in less than 2 per cent of 470 cases. Cornell and Shookhoff in 1944 collected all the cases of actinomycotic involvement of the heart and pericardium and added 3 of their own, bringing the total reported cases to 68. Their study emphasized that actinomycosis usually involves the heart by direct extension. Hematogenous spread is less common. When involvement results from direct extension the pericardium is invaded first, usually with complete obliteration of the pericardial space. The myocardium is commonly involved and not infrequently the process extends to involve and perforate the endocardium. With hematogenous spread the myocardium is the site of primary involvement. Extension to the pericardium or endocardium occurs in only a few such instances.

Sixty of the 68 cases had adequate study. Twenty-three had clinical involvement of the heart or pericardium (see table 2).

Only 4 cases had a pericardial friction rub as the principle manifestations of cardiac disease. All had massive involvement elsewhere and all were fatal. One was due to pyemic involvement and 3 to direct extension.

Certain important clinical facts are brought out by Cornell and Shookhoff in their work: (1) cardiac actinomycosis most commonly results from invasion of the pericardium by direct extension; (2) pyemic involvement of the heart is rare (in such cases the myocardium is first involved); (3) clinical signs of cardiac involvement occur in about 50 per cent of patients with actinomycotic heart disease; and (4) congestive heart failure is the most common clinical manifestation.
Seven additional cases of actinomycotic heart disease were found in the literature. In none was the presenting sign a pericarditis. Three cases resembled subacute bacterial endocarditis. Two were fatal and the diagnosis was established at autopsy. The third was apparently cured by massive doses of penicillin. An "actinomyces-like" organism was grown aerobically from several blood samples and was classified as Actinomyces septicus. The organisms found in the first 2 cases were identified as A. graminis and an "actinomyces" respectively. In 3 cases actinomyces involved the heart by extension from the lungs. All were fatal. The cardiac lesion was constrictive pericarditis. The specific etiologic agent (A. graminis) was identified in one of these cases. One patient died as a result of hypertensive cardiovascular disease and congestive failure. Necropsy revealed mitral valve lesions from which aerobic actinomyces were cultured.

The case presented here is, therefore, unique in having a pericarditis, without massive involvement elsewhere in the body.

**Table 1.**—*Type of Clinical Involvement in Actinomyotic Heart Disease*

<table>
<thead>
<tr>
<th>Clinical Involvement</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>13</td>
</tr>
<tr>
<td>Pericardial friction rub</td>
<td>4</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>2</td>
</tr>
<tr>
<td>Circulatory collapse</td>
<td>2</td>
</tr>
<tr>
<td>Simulating SBE</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac irregularity</td>
<td>1</td>
</tr>
</tbody>
</table>

**Total number cases with clinical heart disease** 23

**Table 2.**—*The Pathogenic Actinomyces*

<table>
<thead>
<tr>
<th>Group</th>
<th>Organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Anaerobic group:</strong></td>
<td></td>
</tr>
<tr>
<td>Actinomyces bovis (hominis, Wolff-Israel)</td>
<td></td>
</tr>
<tr>
<td><strong>B. Aerobic group:</strong></td>
<td></td>
</tr>
<tr>
<td>1. Non-acid Fast:</td>
<td></td>
</tr>
<tr>
<td>a. Actinomyces madurae</td>
<td></td>
</tr>
<tr>
<td>b. Actinomyces graminis</td>
<td></td>
</tr>
<tr>
<td>c. Actinomyces caprae</td>
<td></td>
</tr>
<tr>
<td>d. Actinomyces somaliensis</td>
<td></td>
</tr>
<tr>
<td>2. Acid Fast: (Also called Nocardia)</td>
<td></td>
</tr>
<tr>
<td>a. Actinomyces farcinicus</td>
<td></td>
</tr>
<tr>
<td>b. Actinomyces asteroides</td>
<td></td>
</tr>
<tr>
<td>c. Actinomyces gyposomes</td>
<td></td>
</tr>
<tr>
<td><strong>C. Facultive aerobic group:</strong></td>
<td></td>
</tr>
<tr>
<td>Actinomyces muris (Streptobacillus moniliformis)</td>
<td></td>
</tr>
</tbody>
</table>

**Modern Concepts of Pathogenesis and Treatment**

The term actinomycosis includes infections caused by any member of the group of pathogenic fungi known collectively as actinomyces. The generic name, actinomyces, is applied to all of the forms by Topley and Wilson. However, a small group of the actinomyces, differing from the rest in cultural characteristics, are referred to by other authors as the genus nocardia. A classification of the pathogenic actinomyces, based upon their oxygen requirements, is included in table 2.

The majority of pathogenic actinomyces are anaerobic and take the Gram stain. In the differentiation from the nonpathogenic forms it is important to determine whether the organism is acid fast, aerobic or anaerobic. *Actinomyces bovis*, the causative organism in the majority of human infections, is anaerobic and non-acid fast. *Actinomyces (Nocardia) asteroides*, a common inhabitant of the human mouth is responsible for about 10 per cent of human infections, chiefly those simulating tuberculosis or involving brain or meninges. It is aerobic and acid fast.

*Actinomyces madurae*, *A. farcinicus* and *A. muris* (the causative organisms of Madura foot, farcy and ratbite fever respectively) are usually not included under a discussion of human actinomycosis. The other organisms, *A. graminis*, *A. caprae* and *A. somaliensis* have rarely been reported as the causative organisms of human actinomycosis.

The exogenous theory of infection, postulating contact with animals, animal products, grasses or cereals was accepted in the past. Contact with animals suffering from "lumpy jaw," or a habit of chewing on grasses or straws was felt to be important in initiating an actinomycotic infection. More recent investigations point to an endogenous focus, which becomes active under proper conditions of lowered resistance. The following data support this hypothesis:

1. The majority of actinomyces present on grasses, grains and cereals have proved to be harmless saprophytes. Most common among these is *Actinomyces graminis*.

2. *Actinomyces bovis*, the organism responsi-
ble for the majority of human cases, has been isolated only from the animal body.\textsuperscript{1, 4, 11, 14, 21}

3. The human mouth has been established as the normal habitat of \textit{Actinomyces bovis}. It has been isolated from pyorrhea pus, dental seum, salivary calculi, carious teeth and tonsillar crypts.\textsuperscript{4, 5, 7, 12, 15, 16}

4. Actinomycotic lesions due to \textit{A. bovis} have been reported following the human bite.\textsuperscript{4, 14, 15, 18}

5. Actinomycosis is no more common in rural than urban areas.\textsuperscript{4, 7, 10}

6. There is no evidence of man-to-man or animal-to-man transmission except by the human bite.\textsuperscript{2, 4, 11, 14, 18}

7. Oral sepsis is frequently associated with the development of actinomycosis, and actinomyces have been cultured from the blood immediately after exodontia.\textsuperscript{4, 18}

In the treatment of human actinomycosis the iodides, thymol, irradiation, surgery, sulphonamides, penicillin and aureomycin have been used. At the present time the therapeutic regimen usually consists of the administration of iodides, sulphonamides, penicillin, aureomycin and surgery where indicated.

The use of iodides in human actinomycosis was initiated by Nocard (1885) who found them specific in the treatment of actinomycotic-like lesions in cattle.\textsuperscript{4} Since then they have been widely used in treating the human form of the disease. The organism so successfully treated by Nocard and others was \textit{Actinobacillus lignieresi}, for which iodides are specific.\textsuperscript{1}

It was not an actinomyces. \textit{Actinomyces bovis} has been grown successfully on media containing 2 per cent iodides. At present there seems to be no indication for the use of iodides in human actinomycosis.\textsuperscript{4, 5, 16}

Favorable reports have appeared in the literature regarding the use of sulphonamides,\textsuperscript{27, 28} penicillin,\textsuperscript{5, 7, 11} and surgery,\textsuperscript{29, 30} or combinations of the three.\textsuperscript{4, 30—32, 34} Reports on the potency of penicillin indicate that some strains of actinomyces are more susceptible than others.\textsuperscript{29, 30} The best regimen at present is probably a combination of penicillin and the sulphonamides. Surgical procedures may be necessary to remove pus and dead tissue. The importance of blood transfusions in the debilitated has been stressed by Lyons and others.\textsuperscript{30, 34} Aureomycin in the treatment of cervicofacial actinomycosis was recently reported.\textsuperscript{19} Apparently aureomycin has little effect on the organism and, until further investigation supports the above work, its use is not recommended.\textsuperscript{35}

**Summary**

1. A case of recovered pericarditis caused by \textit{Actinomyces bovis} is presented.

2. The available literature on cardiac actinomycosis is reviewed and summarized.

3. The pathogenesis and modern therapy of human actinomycosis is discussed.

**Acknowledgment**

I am indebted to Dr. Daniel J. Glomset, Chief of the Medical Service, for helpful advice in the preparation of this manuscript.

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Circulation. 1951;3:854-858
doi: 10.1161/01.CIR.3.6.854

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1951 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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